EFIC5-1079

NON-PRESCRIPTION (OVER THE COUNTER) ANALGESICS

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Pain meetings typically concentrate on pain problems seen in primary or secondary care. While pain is a common problem in both these setting, minor pain problems are even more common than that, and are often dealt with by patients themselves using analgesics available without proscription. Use of non-prescription analgesics is common, with several surveys indicating that up to half the population will have used a non-prescription analgesic within the previous two weeks.

This workshop will examine the non-prescription analgesics (oral aspirin, NSAIDs, paracetamol, and topical analgesics) in three ways.

Professor Henry McQuay (Oxford) will chair the workshop and will set the scene for the speakers by outlining the extent of use of non-prescription analgesics. Terry Maguire (Queens University, Belfast) will look at the pharmacy perspective, examining the different regulatory considerations around Europe, and the volume of analgesic purchases and consumption of analgesics by the population as a whole. Angel Lanas (University of Zaragoza) will examine the question of safety of aspirin, NSAIDs, and paracetamol taken intermittently. This will look principally at the gastrointestinal risks, but will touch on other potential risks as well. Andrew Moore (University of Oxford) will present evidence of efficacy of non-prescription analgesics for acute pain conditions, including tension headache and migraine, and strains and sprains for topical products.

EFIC5-1083

EPIDEMIOLOGICAL APPROACHES TO THE ETIOLOGY OF CHRONIC PAIN

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The study of causal mechanisms in pain is commonly thought to be the exclusive province of true experimental designs. However, such studies are also limited by uncertain generalizability to pain occurring in real-world contexts where multiple influences come to bear. By contrast, epidemiological studies have high external validity, but are considered weak ground for causal conclusions. But is this always the case? In this workshop we discuss when and how epidemiological studies can contribute to our understanding of the etiology of chronic pain. First, Professor MacFarlane will discuss to what extent and under which circumstances causal inferences may be drawn from epidemiological studies. He will provide historical examples where valid discovery has been made, and contrast these with research leading to erroneous or exaggerated conclusions. Next, Dr. Nielsen will discuss the application of experimental pain models in large population based studies. He will consider how such data can be used to test and extend findings from studies of non-representative samples, and to provide new insight in areas that cannot be addressed in true experimental designs. Finally, Dr. Williams will discuss how multivariate twin studies can be used to identify shared genetic and environmental etiology across pain conditions, thus providing the foundation for an etiologically based classification of pain. She will also describe how co-twin control designs can be used to test causal hypotheses about environmental influences in pain.

EFIC5-1084

PAIN IN DEMENTIA: THE NEED FOR MULTIDISCIPLINARY AND MULTI-PROFESSIONAL PERSPECTIVES

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Pain and dementia gain equally high prevalence in the oldest old. The combination of the two conditions produces miserable living conditions for the patients and enormous scientific as well as clinical challenges for those providing treatment and care. Valid and reliable pain assessment is still a problem, RCTs for pain in dementia are almost completely missing, pain care in nursing homes cannot be based on empirically scrutinized guidelines, and pain and dementia combined complicate the palliative phase to the risk of further losing dignity. In this situation, a network of experts from 16 European countries (COST TD1005) could be established, searching for solutions to the described problems. One of the early results is the urgent need for multidisciplinary and multi-professional collaborations and perspectives, including geriatricians (also specialized for nursing homes), palliative care physicians, (neuro-)psychologists, and nurses (also specialized for the care of the elderly). The workshop will present the necessary multi-perspective view at the problem of pain in dementia, which has been developed by this European network, and will approach a multidisciplinary and multi-professional audience.

EFIC5-1085

EXPLORING VISCERAL PAIN IN THE 21ST CENTURY

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The goal of this symposium is to provide an updated overview of methods and techniques recently applied in visceral pain based on findings in selected gastrointestinal diseases. An overview of the pain mechanisms involved in organic as well as functional gastrointestinal disorders are provided, with special emphasis on human experimental pain, including quantitative sensory testing, assessment of the associated autonomic responses to visceral pain, advanced neurophysiological methods and modern imagining methods. Pain mechanisms in oesophageal disorders, chronic pancreatitis, diabetic visceral neuropathy and irritable bowel syndrome will be reviewed. This is followed by a discussion of advantages and shortcomings of the different methods and it is discussed how these methods can be combined in future studies of visceral pain to gain a more comprehensive understanding of the involved mechanisms, which again could lead to better treatments.

EFIC5-1086

CARDIAC NOCICEPTION: HOW DO PAIN MODULATION PROFILE AND PERSONALITY AFFECT PAIN EXPERIENCE AND DELAY IN SEEKING MEDICAL HELP

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The inconsistent association between chest pain intensity and the severity of cardiac ischemia manifests in several ways including silent ischemia, silent infarction and delay in seeking medical attention despite acute coronary occlusion. These frequent clinical presentations of aberrant cardiac nociception are of considerable importance because they are associated with increased morbidity and mortality, with afflicted patients not benefiting from a prompt medical intervention. The complexity of chest pain experience, patient's pain interpretation and behavior in situations of cardiac pain will be highlighted in this workshop from the theoretical and clinical perspectives. 1. Nociception activated by heart ischemia and necrosis (Doron Aronson): This talk will present the inflammatory processes activated due to myocardial ischemia and myocardial infarction. These processes are central to cardiac remodeling and adaptation of the injured myocardium. The potential interaction of cardiac inflammation with chest pain experienced during the acute phase of myocardial infarction will be discussed. 2. Psychophysical approach to the investigation of chest pain variability (Michal Granot): This session aims to review the mechanisms of pain modulation that affect chest pain experience in the setting of cardiac ischemia. Special attention will be given to the potential effect of the individual pain modulation profile (as assessed by quantitative sensory testing) on pain variability among cardiac ischemia patients. 3. Pain interpretation and behavior (Geert Crombez): This talk will focus on the role of cognitive, emotional and personality variables on pain response to myocardial ischemia and further elaborate how nociceptive impute can evoke pain that is experienced as "non-harmful".

EFIC5-1087

COLD TEMPERATURE PROCESSING IN THE NORMAL AND DISEASED NERVOUS SYSTEM

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Temperature and pain sensations are important for body integrity and homeostasis and are based on a complex integration of labeled-line inputsCold allodynia can be a severe disabling symptom, which may result in reduction of chemotherapy and predict chronic neuropathy. Also ciguatera, a marine food poisoning, can cause severe cold allodynia. New studies have increased our knowledge about the peripheral and central processing of cold and cold pain sensations in the normal and diseased nervous system and offers new therapeutic opportunities for targeting cold pain pathways. Katharina Zimmermann will give an overview of the current concept of ion channels involved in physiological cold temperature sensing in peripheral nerve endings and show how sodium channel activator toxins like ciguatoxins change sensory nerve excitability to create pathological hypersensitivity to cold in Ciguatera sea food poisoning, which resembles the symptom of cold allodynia in various neuropathic pain states.. Francesca Fardo will present novel results on the time-course and source localization of cold-related activity associated with the activation of A-delta and C fibers in healthy volunteers, as detected by magnetoencephalography (MEG). Further, she will provide an overview of the advantages, methodological issues and potential applications of MEG in the study of cold processing. Nanna Finnerup will present clinical neuropathic pain conditions characterized with cold allodynia and paradoxical heat sensations. She will discuss central integration of cold and warm based on experimental and clinical studies showing changes in the thermal grill illusion and paradoxical heat sensation after sensitizing heat-sensitive neurons and in neuropathic pain patients.

EFIC5-1088

PAIN WITHOUT WORDS: DEVELOPMENTS IN PAIN COMMUNICATION AND APPLICATIONS TO CLINICAL PRACTICE AND RESEARCH

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Effective pain assessment and management usually requires that patients communicate their experiences and that healthcare providers accurately understand and respond. Words provide a wealth of information about subjective pain experiences, but they are not always available. Pain is also communicated through multiple nonverbal channels (face, voice, movement), and our understanding of these processes is well advanced. But important translational issues mean that much of what we know about nonverbal pain communication from experiments is not used to answer questions in clinical practice and research. For example: what contextual factors impact on the interpretation of pain signals? How do we effectively differentiate pain from other expressions, such as fear? What is the relationship between felt and expressed pain? Is it possible to develop objective measures that not only detect pain experiences, but efforts to control pain? The goals of this seminar are to provide a state-of-the-art review of the nature of nonverbal pain communication, and critically consider ways in which this body of knowledge can help clinical practice and research. Dr. Keogh will apply a nonverbal pain framework to understand how and why there are gender differences in pain and analgesia. Dr. Williams will consider the possibilities of automated behaviour detection that can be integrated into pain treatment and self-management. Dr. Kappesser will consider how contemporary techniques to measure pain behaviours in nonverbal pain populations (elderly, neonates) can improve pain assessment in the clinic.

EFIC5-1089

NEW STRATEGIES FOR THE DIAGNOSIS AND THERAPY OF LOW BACK PAIN SUBTYPES: FROM THE LABORATORY TO THE PRACTITIONER

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Low back pain is one of the most frequent pain syndromes which virtually every human being is suffering from one day. In this symposium we intend to give an overview of actual screening tools for the subgrouping of back pain syndromes, special diagnostic approaches as well as tools for the daily routine for the most common entities of low back pain syndromes. In the conclusion we will present a pathway for guiding the practitioner from screening to special diagnostics to the most suitable mechanism based treatment, which should be clinically applicable. Screening tools for the subgrouping of back pain patients (Didier bouhassira, chair): Questionnaires and fast neurological examination for a swift but precise allocation of back pain syndromes will be presented in the first part of the symposium. Bed-side quantitative sensory testing (Maren Reimer): In neuropathic pain conditions a new short form of the quantitative sensory testing (QST) protocol provides promising data which makes bedside QST easily applicable by every practitioner in low back pain patients. Special low back pain syndromes need special diagnostic tools (Philipp Hüllemann, co-chair): Due to the herringbone pattern of radicular dermatomes nerve conduction studies (electroneurography) cannot determine the exact height of damaged nerve routs in radiculopathy patients. MRI-studies regularly fail to detect any nerve compression, although, patients suffer from severe low back pain which radiates to the lower legs. Laser evoked potentials specifically elicit pain and temperature conducting A-delta and C-fibers in radiculopathy patients which grants exact detection of the damaged nerve rout.

EFIC5-1090

THE BIDIRECTIONAL EFFECT OF STRESS ON PAIN: FROM MODELS TO MECHANISMS

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Stress has bidirectional effects on pain; suppression (stress-induced analgesia=SIA) or exacerbation (stress-induced hyperalgesia=SIH) depending on its characteristics. Moreover, stress-related conditions and chronic pain are highly co-morbid. We aim to disentangle the complex relationship between the stress and pain, based on findings from animal models of SIA/SIH, from human experimental stress models, and clinical studies. David Fin: will focus on the role of the endocannabinoid system in discrete brain regions in fear-induced analgesia, and SIH in the stress-hypersensitive Wistar-Kyoto rat strain. The endocannabinoid system plays an important role in affective modulation of pain, results which may inform the development of endocannabinoid-targeted therapeutics for improved treatment of pain, affective disorders, and their co-morbidity. Loren Martin: will present novel models to study SIH/SIA in animals and people. This talk will focus on the role of environmental and social stress in mediating hyperalgesia/analgesia in mice and people and the underlying neurochemical basis. Ruth Defrin will present data on the differential affects of acute stress on pain sensitivity vs. pain modulation, and how elite athletes accustomed to physical and emotional stress respond to acute stress as opposed to controls. Effects of acute vs. chronic stress on pain will be discussed. Herta Flor will focus on the relevance of SIA/SIH for mental and psychophysiological disorders. For example, while fibromyalgia is related to deficient SIA, it is enhanced in PTSD although both groups endorse chronic pain. The potential causes of the differential effects, their relation to chronic pain, and the cannabinoid mediation of SIA will be discussed.

EFIC5-1091

NEW THERAPEUTIC RECOMMENDATIONS FOR NEUROPATHIC PAIN: FROM PHARMACOTHERAPY TO NEUROSTIMULATION

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Evidence based recommendations are important to guide the management of neuropathic pain, which is very difficult to treat. In recent years a number of therapeutic recommendations have appeared for the pharmacotherapy of neuropathic pain as well as for neurostimulation therapy. This workshop will present a state of the art of current guidelines for pharmacotherapy of NP, including the recently updated NeuPSIG recommendations, and for invasive and noninvasive neurostimulation therapy. Nanna Finnerup (Aarthus, Denmark) will present the challenges in meta-analyses of pharmacological treatment in neuropathic pain. Nadine ATTAL (Boulogne-Billancourt, FRANCE) will present the new NEUPSIG recommendations on pharmacological treatment of neuropathic pain. Finally Giorgio Cruccu (Roma, Italy) will present the new EFNS and NeuPSIG recommendations for central neurostimulation for neuropathic pain.

EFIC5-1092

SPREAD OF MUSCULOSKELETAL PAIN- COMMON BUT MISUNDERSTOOD

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Chronic widespread musculoskeletal pain is a major clinical problem. Widespread pain includes syndromes often triggered by a local trauma such as e.g. fibromyalgia, whiplash associated disorders, non-specific low back pain. In the early stage of e.g. osteoarthritic pain this is recognized as a localized pain condition but the persistent pain or hypersensitivity may result in a progression to more regional or even widespread symptoms. It is likely that an initial excitation and sensitisation of nociceptors will cause sufficient nociceptive input to the central pain systems to cause sensitisation of dorsal horn neurons and/or at higher brain centres. Injection of nerve growth factor (NGF) has been proposed as a model for the sensitisation process. Intramuscular injections of NGF in humans induce widespread mechanical hyperalgesia and even facilitate central mechanisms such as temporal summation of pain and referred pain. Basic animal data have shown immediate peripheral sensitisation and hyperexcitability of dorsal horn neurons after some days. Repeated intramuscular NGF injections within days have demonstrated an even more expressed central sensitisation in animals and progressively increased soreness in humans. In widespread pain patients the involvement of peripheral sensitisation is probably not the primary mechanism. However, treatment of myofascial trigger points in fibromyalgia patients revealed less widespread pain indicating the importance of pain generators in widespread pain conditions. This topical workshop will translate findings from basic animal (Mense) and human (Graven-Nielsen) studies to clinical findings (Giamberardino) and further demonstrate the importance of the peripheral drive for central mechanisms relevant for widespread pain conditions.

EFIC5-1093

CHRONIC REFRACTORY HEADACHES, BEHIND THE CLINICAL DEFINITION

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The principal task that still lies ahead of physicians treating headache patients in their daily practice is to frame refractory headaches in an updated nosography and treatment. The disability burden and social costs caused by this subset of headache disorders is huge and perpetual. Without a standardized definition of refractory headaches, various unproved medical and surgical practices have been applied to this subset of patients, with results still too scanty and often with weak scientific background. In fact the lack of scientific evidence in defining and validating the concept of refractoriness has prevented its inclusion in the 2013 ICHD-3? Headache Classification. The EFIC Task Force on headache offers this proposal based on recently published Consensus Statements on headache refractoriness and on the rising need of a multidisciplinary approach in chronic headache disorders. The concept of multidisciplinarity also will be further investigated in the course of this Topical Seminar, building a bridge between refractory headaches definition and the broader concept of chronic pain treatment.

EFIC5-1094

FIRST DO NO HARM, POTENTIAL MISUSE OF PAIN MEDICATIONS: OPIOIDS AND GABAPENTINOIDS

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The potential for misuse of prescribed medications for pain has been a prompt for both a study of risks of misuse and for production of guidelines outlining precautions in prescribing. At a societal level, the scale of medication related harms has led to a number of policy initiatives and legislative changes to address the public health problem of prescription medication misuse. The literature to date has focused almost wholly on risks associated with prescription opioids but there are substantial emerging concerns regarding misuse and harms of gabapentin and pregabalin. These drugs have for some time been recognised as problematic by those working with high risk groups including healthcare professionals in custodial settings and workers in addiction and recovery services. The potential for misuse of analgesics other than opioids and the potentially fatal consequences of misuse of analgesic drugs in combination is now properly a concern for all prescribers of both opioids and gabapentin/ pregabalin. This session will start with an overview of the information that prescribers can draw on to make safe and effective prescribing decisions. Current data on harms associated with both opioids and gabapentin/pregabalin will be reviewed. The session will conclude with a description of strategies already in place to minimise harms of prescribed analgesics and will consider some general underpinning messages regarding pharmacotherapy of pain to ensure patients and others are not harmed by use or misuse of analgesic drugs.

EFIC5-1095

INVESTIGATING EXTREME PHENOTYPES OF NEUROPATHIC PAIN: A NEW APPROACH TO UNDERSTAND HOW MECHANISMS OF DISEASE TRANSLATE INTO SPECIFIC CLINICAL SOMATOSENSORY PROFILES

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The clinical presentation of neuropathic pain consists of a heterogeneous mix of sensory gains and deficits, which depend on the mechanisms involved. The somatosensory profile of patients can be determined by using questionnaires and quantitative sensory testing. In this workshop we will discuss the results of studies investigating rare causes of neuropathic pain with specific mechanisms of disease. The causes of neuropathy of the different cohorts investigated are infrequent but serve as 'human models' of particular physiopathology. This gives us an exceptional opportunity to observe how mechanisms of disease are translated into somatosensory profiles. Professor Andrew Rice will show the results on a cohort of Indian leprosy patients with a unique sensory profile where cold, heat, and mechanical detection thresholds are lost but vibration and pressure pain thresholds are preserved. Professor David Bennett will show that VGKC antibodies can induce chronic pain in patients and this can be replicated by injecting the patient's serum in mice, giving the opportunity to test the mechanism in an animal model. Dr Andreas Themistocleous will describe how non-freezing cold induced injury causes a small fibre neuropathy complicated by chronic neuropathic pain. Dr Margarita Calvo will show her data on patients with Epidermolysis Bullosa, a skin disease that leads to degeneration of intraepidermal nerve fibres and to neuropathic pain with an exclusive loss of thermal thresholds. We believe that an effort on understanding the mechanisms behind a particular combination of symptoms and signs is imperative and could lead to a personalised treatment of neuropathic pain.

EFIC5-1096

PAIN IN WHIPLASH ASSOCIATED DISORDERED: THE ROLE OF CENTRAL NERVOUS SYSTEM DYSFUNCTIONS AND EXERCISE THERAPY

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Up to 50% of patients with acute whiplash associated disorders (WAD) do not fully recover and continue experiencing multiple symptoms such as chronic neck pain, fatigue, dizziness, concentration difficulties and headaches. Radiological findings or cervical dysfunctions do not account for the development of chronic WAD. On the other hand, there is increasing evidence that WAD are characterized by dysfunctional stress response systems and hyperexcitability of the central nervous system. The former include a dysfunctional hypothalamus-pituitary-adrenal axis and altered autonomic reactivity, and the latter implies dysfunctional endogenous analgesia (in response to nociceptive stimuli and exercise), decreased spinal reflex thresholds, increased temporal summation of pain, and widespread hyperalgesia. This workshop will not only review and update our understanding of these central nervous dysfunctions in patients with WAD, it will also show that they are interconnected and possibly influenced by exercise (therapy). Indeed, the stress response system can influence pain through several neurophysiologic mechanisms. This notion is supported by the finding that sympathetic nervous system activation has a predictive value in the transition from acute to chronic WAD. Severe stress also leads to diminished availability of several key central nervous system neurotransmitters (e.g. GABA and serotonin), possibly explaining the inability of WAD patients to activate top-down endogenous analgesia (in response to exercise). The workshop will not only explain these mechanisms, it will also address the role of genetic factors and present new findings regarding the role of therapeutic targeting of brain neurotransmitters for activating endogenous analgesia during exercise in people with chronic WAD.

EFIC5-1097

EXPLORING THE RELATIONSHIP BETWEEN PAIN AND COGNITION: PROPERTIES, MECHANISMS AND IMPLICATIONS IN ACUTE AND CHRONIC PAIN

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Alongside sensory and emotional experiences, our contemporary understanding of pain has to also include cognitive processes. For example, attention and learning are not only known to play critical roles in the perception and experience of pain, but cognition is also relevant to our understanding of pain management and issues around analgesia, including distraction and placebo responses. Whilst there have been numerous advances in our understanding of the links between pain and cognition, there are also challenges, such as: the reciprocal nature of pain and cognition (effect of pain on cognition, effect of cognition on pain); how the relationship between pain and cognition may varies across different types of pain; as well as how such insights can be directly applied to practice. Therefore, the goals of this seminar are to consider the relationship between cognition and pain, as well as the translational issues around knowledge and practice. Dr. Keogh will consider transitions between laboratory-induced pain and everyday pain (headache, menstrual pain), as well as look at methods of recruitment and testing of cognition and pain. Dr. Lautenbacher will consider the novel application of attentional biases towards threat in predicting post-surgical pain. Dr. Bingel will consider the neural basis of the effects of cognition on pain, pain relief and analgesic treatments. Collectively, the seminar will provide attendees with a review of the current evidence base for the relationship between cognition and pain, consider properties and mechanisms, identify current/new challenges, as well as considering potential applications.

EFIC5-1098

INFANT PAIN ASSESSMENT IN CLINICAL PRACTICE: CURRENT TRENDS AND FUTURE DIRECTIONS

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Assessing pain in infants is a challenge as they are not able to verbally communicate their pain. Therefore, infants are wholly dependent on caregivers (parents, clinicians) to acknowledge and appropriately assess their pain signalling. Infants, particularly premature neonates, are subjected to a large number of painful events and repeated neonatal pain experiences can lead to severe long-term consequences. Adequate management of infant pain is predicated on a foundation of timely and accurate assessment. Fortunately, there are a number of tools available today to assess infant pain based on behavioral, physiological, and contextual cues. However, they vary in their psychometric evidence base and their utility in clinical settings. When not using a standardized assessment tool, clinicians implicitly and subjectively extract from a diversity of cues those indicative of pain. Although very common, still only little is known about determinants of this process. This multidisciplinary seminar synergizes the international expertise of three infant pain experts. Its goals are to (1) highlight strengths and weaknesses of existing pain assessment tools, (2) provide an overview of subjective pain estimations' determinants and (3) suggest possible ways to improve the gap between research on pain assessment and its practical application. Accordingly, Dr. Kappesser (Psychology; Germany) will talk about a systematic comparison of the most widely used tools; Dr. Pillai Riddell (Psychology; Canada) will talk about determinants of subjective measures of infant pain; and Dr. Axelin (Nursing; Finland) will talk about the implementation of assessment tools in clinical practice and parents' role in infant pain assessment.

EFIC5-1099

MECHANISMS OF PAIN VULNERABILITY: WHY DO ONLY SOME OF US HAVE TO SUFFER?

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It is well known that there are many risk factors for chronic pain. We propose to hold a seminar that will introduce attendees to the concept of pain vulnerability and its potential biological underpinnings. We will give them a brief, but cutting edge overview of two very important and exciting areas of research: genetics and epigenetics. We will review how rapidly evolving technology platforms are being applied to both experimental models and also clinical pain states.

Professor Henrik Kehlet, a renowned Professor of Surgery and Perioperative Pain, will introduce the audience to the concept of chronic pain vulnerability. Using the extensive epidemiological data he has collected over the years, he will explain who is most at risk of developing persistent pain and how this knowledge is already being applied in the clinic.
Professor David Bennett, an expert in the genetic basis of inherited painful neuropathies and channelopathies, will give attendees an idea of how high impact gene mutations and variants are involved in the emergence of chronic pain.
Dr Franziska Denk will introduce epigenetics as a mechanism that could influence pain risk, with early life events and the environment potentially able to produce longstanding changes in chromatin structure and function.

In our seminar, we want to deliver knowledge in a way that is intriguing and useful not only to basic scientists, but also to the many clinicians, nurses and other healthcare professionals that may be in attendance. We have therefore made an effort to employ a multidisciplinary approach.

EFIC5-1100

ABNORMALITIES OF THE SENSORY PROCESSING OF PAIN: WHAT CAN WE DO ABOUT IT?

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Hyperalgesia and allodynia have received great attention because of their frequent presence in both acute and chronic pain conditions, including neuropathic, inflammatory and even idiopathic pains. Understanding these sensory changes will require translating mechanisms in animal models to guide potential treatments in patients. Sensory testing, intensities and areas of pain can be used, both in patients and in animals, to study neuronal function. The aim of this workshop is to translate backwards and forwards by presenting current knowledge of underlying mechanisms for enhanced pain sensations from the pre-clinical and patient domains. Underlying events could include changes in peripheral processes caused by nerve or tissue damage as well as important plasticity in central pain transmission and modulatory circuits ranging from the spinal cord to the brain and back again. Professors Jensen and Dickenson will present data on potential mechanisms, both peripheral and central, and relate to present and future treatments for enhanced pain states. Emphasis will be on sub-dividing patients with neuropathic pain and this will be discussed in terms of predictive values of animal models. However opioid induced hyperalgesia (OIH) appears to be a phenomenon of central excitatory opposing changes leading to hyperalgesia with a normal periphery. Junior researcher Dr Bannister, and Professor Baron will discuss aspects of OIH in terms of mechanisms and implications for patient care with those receiving strong opioid analgesics.

EFIC5-1101

"SENSORY PROFILES" IN CHRONIC PAIN: FROM SYMPTOMS TO MECHANISM AND POSSIBLE TO TARGETED TREATMENT

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Patients suffering from neuropathic pain have different sensory abnormalities on clinical examination and describe experiencing pain of diverse types, some spontaneous and others provoked. Although in some etiologic categories specific types of pain may predominate, none of them are etiologic specific. Classifying neuropathic pain according to a symptom-based rather than an aetiology-based approach might minimize pathophysiological heterogeneity within the groups under study and thus help in targeting therapy to the individual patient. It would also be useful in testing new drugs. In recent years, increased combination of newly proposed screening questionnaires, and diagnostic procedures such as quantitative sensory testing, pain-related evoked potentials and skin biopsy, have therefore advanced the mechanistic approach to pain management leading to the development of the so-called sensory profiles. In this workshop the first presentation (M Haanpaa) is dedicated to the theory of sensory profiles and how clinical assessment supports the usefulness of a symptom-based classification of neuropathic pain. The second presentation (A Truini) will deal with the neurophysyological and skin biopsy evidence showing that a specific type of pain is mediated by a distinct underlying mechanism. The third presentation (D Bouhassira) aims at showing how a symptom-based classification of neuropathic pain might improve the possibility of detecting effective drugs in clinical trials. In particular this presentation will show how a series of drug trials that failed to meet the primary endpoint, would have been successful by restricting the analysis to patients with specific types of pain.

EFIC5-1102

LEARNING PAIN-RELATED FEAR: NEURAL MECHANISMS, GENERALIZATION AND EXTINCTION

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Converging evidence supports the role of associative fear learning in the development, maintenance, and treatment of chronic pain. However, neural mechanisms underlying pain-related fear memory formation and extinction are only beginning to be understood. Novel behavioral studies on the acquisition, generalization, and extinction of pain-related fear are emerging. Sigrid Elsenbruch will highlight neural mechanisms underlying pain-related fear memory formation and extinction. She will discuss brain imaging evidence among healthy individuals and patients with chronic pain, with a particular focus on visceral pain and irritable bowel syndrome. Nils Niederstrasser will present recent research on generalization of pain-related fear and the associated role of executive functions. He will discuss novel research examining fear acquisition using a proprioceptive fear-conditioning paradigm with joystick movements and generalization to novel movements with varying similarity to conditioned movements. Results on self-reports, eyeblink startle responses, and executive functions will be discussed. Brjánn Ljótsson will present a series of studies on exposure-based cognitive behavioral treatment delivered over the internet. The treatment includes exposure to IBS-symptoms and other fear-related contexts and brief mindfulness training. The treatment has been evaluated in four clinical trials with response rates between 59% and 65%. Zina Trost will discuss application of virtual reality (VR) gaming technologies to facilitate graded-exposure treatment for high-fear individuals disabled by chronic pain. She will discuss recent data from feasibility research examining development of a VR graded-exposure intervention to reduce disability among high-fear individuals with chronic low back pain, including qualitative feedback from patients regarding this potential therapy.

EFIC5-1103

COPING WITH PAIN IN PARENTS AND THEIR CHILDREN WITH CHRONIC PAIN

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Parents are important in learning how to handle pain: the way adults with chronic pain will deal with their own pain can influence their child's way of coping with future pain complaints. And in addition, the way a parent respond to his/her child in pain is important to how both parent and child attempt to cope with pain. During recent years, several studies have addressed interaction between parents and their children related to chronic pain by studying parental modelling and parental reinforcement of pain behaviours. The main aim of the proposed symposium is to present the current state of the art on parent-child interaction related to children and adolescents with chronic pain. A growing body of research points to the key role of family and parent factors in understanding parental responses. In this symposium, first, results of experimental studies on explaining parental responses to child pain are presented. The effect that catastrophizing and family functioning will have on parent-child interaction will be explored. Second, we will discuss potential short and long term changes in parental pain related behaviour as a result of a multidisciplinary rehabilitation program with special attention for parents. Finally, promising treatment modalities to assist parents in helping their child (and the family) to cope with chronic pain will be addressed. The content of the parent program of an out-patient (the Netherlands) and in-patient rehabilitation program (Germany) for children with chronic pain will be discussed. In this symposium evidence on theoretical models, assessment, and treatment will be integrated.

EFIC5-1104

PAIN AND SUICIDE: A SILENT EPIDEMIC

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Death by suicide has become a global epidemic. Every 40 seconds someone in the world dies of suicide. An estimated 804,000 suicide deaths occurred worldwide in 2012. Individuals with chronic pain commonly have significant concomitant psychiatric and medical disorders placing them at higher risk for suicide with the prevalence of suicidal ideation in this patient population ranging from 19% to greater than 50%. One recent systematic review revealed that the risk of successful suicide was doubled in patients with chronic pain as compared to non-pain controls. This multidisciplinary, international panel will review the current literature on the epidemiology of suicidal ideation in the pain population and discuss assessing suicide in the pain population and modifiable mediators of pain and suicide.

EFIC5-1105

CORTICAL STIMULATION FOR PAIN CONTROL: WHERE DO WE STAND? WHAT DO WE DO NEXT?

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Electromagnetic stimulation of the motor cortex for the relief of neuropathic pain has been used for more than 20 years, with appreciable success. Work in pain patients, healthy humans and animal models have provided a consistent corpus of data on the mechanisms most likely to underlie the clinical effects of this therapy, and thus optimise patient selection. In parallel, continuous investigation of pain cortical processing has provided insights into new possible cortical targets to modulate clinical pain. In this workshop we will present (a) the current status of the invasive and non-invasive techniques of cortical stimulation, as they are applied in clinical practice; (b) the insights on the underlying mechanisms gained from both animal models and clinical research; (c) the limits and current challenges to improve clinical efficacy, and (d) the opening vistas to new possible cortical targets using invasive and non-invasive techniques.

EFIC5-1106

Experience and expectation modulate placebo effects in patients

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Placebo effects have been widely studied and there have been huge steps forward to identify the underlying mechanisms. Expectancy and classical conditioning have been identified as the main factors. Expectancy is thought to generate the placebo response via expectations regarding the 'treatment'. It is introduced via verbal instruction (e.g. 'powerful painkiller'), while the deliberate reduction of painful stimuli after administration of a placebo is often referred to as a 'classical conditioning'. Recent studies have shown that especially contextual factors influence the magnitude of the placebo response. They can serve as cues to remind subjects of prior experience and can thus influence not only expectations but also classical conditioning. Considering that learning, expectancy and contextual factors are important; it is conceivable that patients differ from controls due to their circumstances. However, research in patients is sparse but urgently needed. This symposium thus focuses on contextual factors that influence placebo responses and places a large emphasis on investigating patients. We will not only focus on presenting scientific data but also highlight how the results can be transferred into practice. Regine Klinger will disentangle the effects of experience, expectation and attitude on placebo response in chronic pain. Sandra Kamping will investigate the neuronal mechanisms of placebo effects in chronic pain and delineate similarities and differences to controls. Lene Vase will outline psychological and neurophysiological mechanisms underlying placebo effects in chronic neuropathic pain patients. Elisa Frisaldi will show which neurochemical systems can be activated by different contexts, modulating both pain experience and therapeutic response.

EFIC5-1107

The next generation of fear-avoidance models for chronic pain

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The importance of psychological factors in the development and maintenance of chronic pain and associated disability is now virtually established. For the past 20 years the fear-avoidance model has been enormously successful in guiding psychological research into chronic pain and contributing to this understanding. However, accumulating research supports an expansion of the fear-avoidance model, as well as the usefulness of other psychologically-based models of chronic pain that are not principally focused on fear. The goal of this session is to provide an update on recent theoretical developments and evidence in our understanding of psychologically-based models of chronic pain. The first presentation by Professor Crombez will review the challenges of the fear-avoidance models and how these can be addressed from a goal and self-regulation perspective. The second presentation by Professor Peters will discuss chronic pain from the perspective of positive psychology, with an emphasis on shifting focus from vulnerability factors to resilience. The final presentation by Dr Scott will provide an overview of the psychological flexibility model of chronic pain, with an emphasis on recent clinical data supporting components of the model and treatment developments stemming from the model.

EFIC5-1108

Advances in the care of patients with complex regional pain syndrome

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Presenters will review recent advances for the clinical management of patients with CRPS, and explain laboratory data, which underpin them. Prof. Frank Huygen summarizes data on the use novel neuro-stimulation methods in patients with CRPS, including dorsal root ganglion stimulation, high-frequency- and burst stimulation, and external stimulation technologies. A clinical treatment algorithm including these new methods will be proposed (20minutes). Dr. Andreas Goebel will discuss the emerging concepts of autoimmunity and neuro-immune activation in CRPS. He will review results from trials on immune modulating treatments, and outline the future role for such treatments in both short-term, and longstanding CRPS (20minutes). Dr. Valeria Tekus will detail her recently-published passive-transfer-trauma model for CRPS, in which the transfer of CRPS-patient-IgG elicits relevant CRPS signs in the animals; she will also review novel data in an enhanced model, and discuss implications for future CRPS research (10minutes). Both early Diagnosis of-, and appropriate care for patients with CRPS is crucially dependent on the involvement of Primary Care. Patients often feel abandoned by their primary care physician, whom they perceive to have insufficient understanding about CRPS. Dr. Chris Barker will summarize European CRPS Guideline documents on this topic, and delineate an emerging model of shared care between primary and secondary care.

EFIC5-1109

Approach to activity engagement in musculoskeletal pain- translation from research to clinical application

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The recommendation to return to normal activity in daily life as soon as possible is a central part of international guidelines on the management of acute musculoskeletal pain. Furthermore, physical exercise is a key factor in the treatment of chronic musculoskeletal pain. Conversely, physical inactivity on the one hand, and physical overactivity on the other are shown as risk factors for chronic pain. Understanding the mechanisms by which physical activity can modulate pain sensitivity and how individual pain-response patterns of activity approach can modulate pain and physical function will help to disentangle these conflicting findings and present a translational perspective on the role of physical activity in pain prevention and management. In this topical seminar we will highlight basic research in rodents and humans as well as human studies in healthy people and in patients with musculoskeletal pain in order to unravel the complex relationship between pain, physical activity and physical function. In two presentations we will present recent research on assessment and characterization of the musculoskeletal pain sensitivity following exercise in healthy subjects (T. Graven-Nielsen) and in patients with various kinds of chronic pain, i.e., rheumatoid arthritis, osteoarthritis and fibromyalgia (E. Kosek). Next, we will highlight the importance of different kinds of behavioral approach to physical activity in daily life elucidating especially the role of pain-related avoidance versus endurance behavior (M. Hasenbring). Finally, our young researcher, E. Fehrmann, will present results on the role of subgrouping patients based on the avoidance-endurance model for their pain, function and physical performance.

EFIC5-1110

Chronic pain modulation by neuroinflammation

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The CNS is endowed with an elaborated response repertoire termed 'neuroinflammation', which enables it to cope with pathogens, toxins, traumata and degeneration. On the basis of recent publications, we deduce that orchestrated actions of immune cells, vascular cells and neurons that constitute neuroinflammation are not only provoked by pathological conditions within the CNS, but can also be triggered by noxious stimuli or opioids. Effective triggers for neuroinflammation in the brain and / or the spinal cord include peripheral neuropathies, continued opioid application or abrupt withdrawal from opioids and increased activity in nociceptive nerve fibres, e.g. during peripheral trauma or inflammation. The latter form of activity-driven neuroinflammation has been termed "neurogenic neuroinflammation". In this workshop we will present latest developments in the genesis of neuroinflammation, its impact on nociceptive processing in the spinal cord and brain and clinical implications for the prevention and the treatment of chronic pain conditions.

EFIC5-1113

Painful diabetic neuropathy: from pathophysiology to therapy

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Diabetes is increasingly common, currently more than 60 mil Europeans have diabetes. Of these 50% will develop neuropathy and half of these corresponding up to 15 mil Europeans will eventually develop painful diabetic neuropathy (PDN). The pathophysiology of PDN is unknown. Researchers have either favored a metabolic mechanism where oxidative stress causes nerve damage or a microvascular etiology giving rise to ischemia and nerve pain injury. It is unclear why some patients with neuropathy develop pain and others not. In this workshop we will present novel findings on pathophysiology, distinguishing features between painful and non-painful neuropathy and how this may possibly influence the therapy of PDN. Prof Leif Ostergaard, Denmark will present a new hypothesis in which endoneural hypoxia may be the culprit for the nerve damage using two-photon microscophy and optical coherence tomography to demonstrate possible nerve tissue hypoxia. Prof David Bennett, UK will discuss similarities and differences between painful and non-painful diabetic neuropathy based on questionnaires, clinical profiling and skin nerve fiber pathology. Junior Doctor Silvia La Cesa will discuss the diagnostic assessemnt of painful and painless diabetic neuropathy. Finally Prof Troels S. Jensen will present a rational based pharmacotherapy based on existing evidence and proposed novel pathophysiology.

EFIC5-1115

Improving pain research and pain treatments by improving classification: The ICD-11 proposal for the classification of chronic pain

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The classification of chronic pain conditions in ICD-10 was unsatisfactory. Therefore IASP established a working group to develop a new classification system, which should be fed into the ICD-11 developmental process. The World Health Organization approved this working group, and encouraged it to develop a virtual ICD-11 chapter on chronic pain conditions. Meanwhile a proposal has been finalized that is in accordance with ICD-11 principles, but also practical and feasible e.g. for primary care needs. It is suggested to consider seven main categories of chronic pain in this system: chronic primary pain; chronic cancer pain; chronic postsurgical and posttraumatic pain; chronic neuropathic pain; chronic headache and orofacial pain; chronic visceral pain; chronic musculoskeletal pain. This proposal will be presented and discussed with the participants. Most critical aspects are whether this proposal helps not only to improve diagnostic and therapeutic decisions for pain patients in health care systems, but also to facilitate the installation of pain research programs, and to demonstrate the burden of chronic pain conditions to stakeholders in politics.